

EXHIBIT 7



The heritability fallacy

David S. Moore^{1*} and David Shenk²

The term ‘heritability,’ as it is used today in human behavioral genetics, is one of the most misleading in the history of science. Contrary to popular belief, the measurable heritability of a trait *does not* tell us how ‘genetically inheritable’ that trait is. Further, it does not inform us about what causes a trait, the relative influence of genes in the development of a trait, or the relative influence of the environment in the development of a trait. Because we already know that genetic factors have significant influence on the development of *all* human traits, measures of heritability are of little value, except in very rare cases. We, therefore, suggest that continued use of the term does enormous damage to the public understanding of how human beings develop their individual traits and identities.

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INTRODUCTION

If someone were to tell you that research has proven that human intelligence is highly ‘heritable,’ what would you think that means? Most people would probably assume that it means people inherit a significant percentage of their intelligence directly, via their parents’ genes. In fact, though, the scientific terms ‘heritable’ and ‘heritability’ actually have very little to do with genetic inheritance. This is confusing, because ‘heritability’ sounds like it means the same thing as ‘inheritability.’ The confusion about what ‘heritability’ actually measures significantly adds to a deep misunderstanding about how, exactly, our genomes contribute to our observable characteristics (see Charney, Genes, behavior, and behavior genetics, *WIREs Cogn Sci*, also in the collection *How We Develop*).

THE APPROPRIATION OF ‘HERITABLE’

For hundreds of years, the word ‘heritable’ was used without confusion as a synonym for ‘hereditary.’ But in the early 20th century, the word was repurposed to

represent something new and rather narrow. At that time, geneticists had a strictly deterministic understanding of how genes influence the formation of traits. They considered the relationship between genes and the environment to be akin to the relationship between a plant seed and the rain that waters it: Genes were thought to contain specific, blueprint-like instructions for the formation of traits, whereas the environment provided the nutrients and other salubrious conditions that would allow those instructions to unfold. According to this earlier way of thinking, a person’s DNA has specific instructions for blue eyes, or athletic arms, or a mathematical mind; the environment merely allows for emphasis or de-emphasis of those already-designed traits. (If this sounds familiar, it’s probably because it strongly resembles what many of us were taught about genetics in grade school.)

The term heritability was first given this new meaning in J. L. Lush’s 1937 book *Animal Breeding Plans*.¹ In that text, Lush proposed a calculation for what he called ‘heritability’ that neatly codified the then-popular deterministic viewpoint. Because, Lush argued, an animal’s *phenotype* (i.e., its observable traits, such as intelligence, height, eye color, etc.) is a function of genetic instructions *plus* the finishing influence of the environment, we should be able to statistically separate the influence of each.² Relying on mathematical guidelines from the geneticist Sewall Wright, Lush proposed that in any given group:

$$V_p \text{ (phenotypic variation)} = V_g \text{ (genetic variation)} + V_e \text{ (environmental variation)}$$

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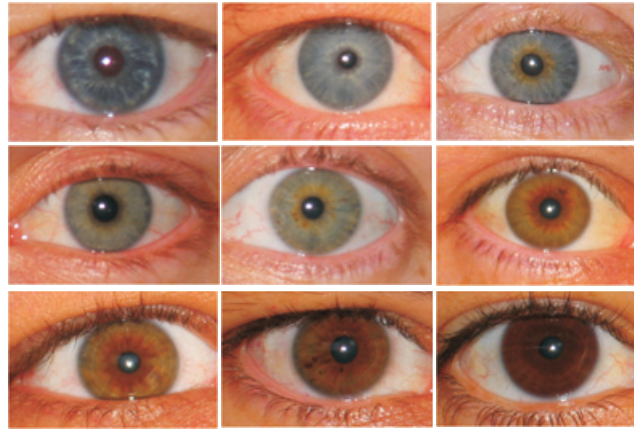


FIGURE 1 | Natural variability in human eye colors. Source: Sturm and Frudakis.³

Lush asserted that the V_g portion of that total can reasonably be termed ‘heritability,’ as it revealed the portion of the trait variation that could be accounted for by variation in genes. The intention was ‘to quantify the level of predictability of passage of a biologically interesting phenotype from parent to offspring’. In this way, the new technical use of ‘heritability’ accurately reflected that period’s understanding of genetic determinism. Still, it was a curious appropriation of the term, because—even by the admission of its proponents—it was meant only to represent how *variation* in DNA relates to *variation* in traits across a population, not to be a measure of the actual influence of genes on the development of any given trait. For example, in a large group of people with eyes of different colors (Figure 1), ‘ V_g ’ only represents the extent to which *variation* in the group’s DNA accounts for variation in different eye colors *in that group*—not whether or how DNA is responsible for the development of eye color. In that sense, it was a highly misleading new use of the term (even in the context of determinism) that was bound to cause confusion: And indeed it did.

TWIN STUDIES

The new use of the term caught on. Since that time, behavioral geneticists have conducted hundreds of studies derived from the Lush-Wright concept of heritability. The most prominent method to determine heritability in human beings has been statistical comparisons of identical and fraternal twins. In what *seems* like a straightforward approach, researchers compute correlations for a trait among identical twins and compare

them to correlations for that trait among fraternal twins. Because identical twins share 100% of their DNA and fraternal twins share, on average, only 50% of their DNA, this statistical comparison yields a crisp number that *seems* to highlight that portion of a trait *caused* by genetic instruction. Even though behavioral geneticists are sometimes careful to point out that each ‘heritability’ measure only actually applies to variations in a specific group, they often use the term ‘heritability’ in a way that conveys a sense of direct genetic influence on traits. Also, because the term (if not its new-fangled scientific meaning) is so familiar to the public, the casual misinterpretation of the term’s narrower meaning has been rampant in the popular press. For many decades now, we have been entertained with journalistic accounts of twin studies suggesting that ‘personality is heritable’,⁴ ‘criminals are born, not made’,⁵ and ‘cheating genes play [a] large role in female infidelity’.⁶ Twin studies have reaffirmed the strong public impression that some physical and personality traits can be passed directly from parent to child through DNA. While understandable, this impression is flatly incorrect, as brightly illustrated by three significant flaws in some scientists’ use of—and thus the public’s understanding of—the term ‘heritability.’

THE GROUP VS. INDIVIDUAL FLAW

Although the original motivation for the concept of heritability was ‘to quantify the level of predictability of passage of a biologically interesting phenotype from parent to offspring’,² it is essential to realize that the equation $V_p = V_g + V_e$ has no relevance at all when it comes to individual development. *The V_p in that equation refers to variation*

across individuals in a population, not to causal processes that occur within any individual person. Although the term ‘heritability’ is often misunderstood as speaking to the degree to which a certain trait’s appearance in an individual is caused by genetic factors, the logic underlying the equation nullifies that conceptualization. ‘Heritability,’ explains author Matt Ridley, ‘is a slippery concept, much misunderstood. For a start, it is a population average, meaningless for any individual person: you cannot say that Hermia has more heritable intelligence than Helena. When somebody says that heritability of height is 90 percent, he does not and cannot mean that 90 percent of my inches come from genes and 10 percent from my food.’ Instead, he means that 90% of the variation in height in a group of people can be accounted for, statistically, by variations in those people’s DNA. But there is no sense in which the specific DNA variations are causing 90% of a person’s height. As Ridley notes, ‘there is no heritability in height for the individual.’⁷

This group/individual distinction might seem like a mere mathematical technicality, but it is not. Failing to recognize the distinction introduces a critical logical flaw that, on its own, completely undermines the broad popular understanding of the term ‘heritability.’

To understand the profundity of this logical flaw, consider an illustration having nothing to do with biology: One winter, in a particular neighborhood, there is a rash of house fires. Committed to fixing the problem, the city sets out to determine what caused the fires. They gather as much data as they can about all the homes in this fire-ravaged neighborhood—including those that had fires and those that did not. They find that 100% of the fire variation in the group is attributable to whether or not space heaters were present in the various homes.

Question: Is the cause of each individual fire due *only* to the presence of a space heater?

An equation analogous to $V_p = V_g + V_e$ might, if applied to *individual* house fires, seem to suggest that the answer is a resounding yes, because the presence or absence of a space heater accounted for *all* of the variation in house fires in the neighborhood. But, actually, the answer is a resounding no, because as it turns out, every single home in this particular neighborhood was built out of highly combustible wood and painted with highly flammable paint. Importantly, that fact might not have emerged in the investigation

because there was *zero variation* in paint and construction materials within this neighborhood. A statistical investigation focused on variation rather than causation is, if not very narrowly interpreted, likely to lead to a mistaken conclusion about what caused the fires and about what can be done to prevent them in the future.

To see this, consider a newer neighborhood right across the river in which every single home was built with non-flammable aluminum and painted with fire-retardant paint. Even if this neighborhood has just as many space heaters, not a single home here could ever catch fire.

So what causes individual fires? The answer is complex, and statistical variation surveys cannot address the question effectively; although all of the *variation* in fires in the fire-ravaged neighborhood can be accounted for by focusing on space heaters alone, the fact remains that multiple factors—flammable building materials, the presence of heat sources, and available oxygen, for example—are responsible for *collectively* causing fires. Thus, focusing on *variation* rather than causation can contribute to a misleading sense of how important a particular factor might be in contributing to a particular outcome.

A similar argument was advanced decades ago by Richard Lewontin,⁸ who suggested a “thought experiment” that illustrates how even if a human characteristic is 100% heritable, it is still possible to easily influence the development of that characteristic by raising children in a different environment. Lewontin asked us to imagine that we plant a handful of normal seeds in an environmental context in which each developing plant receives the identical amount of light and the identical amount of nutrients, and furthermore, that the amount of light and nutrients each plant receives is adequate to support normal growth (see the left side of Figure 2). In this case, the variation in height that we ultimately see in these plants can be accounted for by referencing genetic variation alone (because all of the plants are grown in the same environment, meaning there is no environmental variation at all); in this situation, the heritability of height would be calculated to be 100%. Next, imagine that we plant another handful of normal seeds in an environmental context in which each developing plant again receives the identical amount of light and the identical amount of nutrients—but now imagine that each plant is provided with a less-than-adequate amount of light and nutrients (see the right side of Figure 2). In this situation, too, the heritability of height

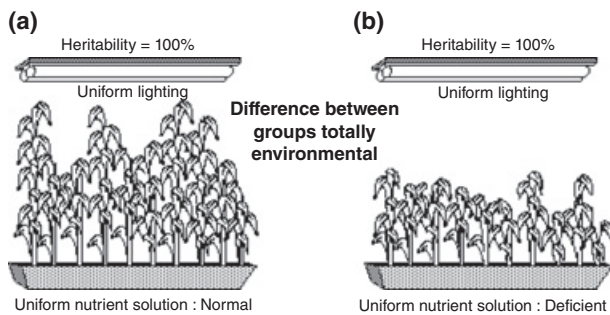


FIGURE 2 | Lewontin's thought experiment. Genetically variable seeds that develop in controlled environments grow to varying heights. The heritability of height in both the (a) and (b) panels is 100%, because all plants in each panel are exposed to the same environment; thus, all of the variation in height (within a panel) is accounted for by *genetic* variation. Despite height being 100% heritable in both the left panel *and* the right panel, plants' heights are still influenced by the quality of the nutrients they encounter in their environments; mature plants that develop in a deficient nutrient solution (b) are shorter, on average, than are mature plants that develop in a normal nutrient solution (a). Source: <http://www.nyu.edu/gsas/dept/philo/faculty/block/papers/plants.gif> and Lewontin.⁹

would be calculated to be 100%, because again, we have carefully controlled the environment in which this second group of plants has grown, so all of the variation in the plants' heights can be accounted for by referencing genetic variation alone. However, even though heritability in each of these subpopulations would be calculated to be 100%, we would still find that on average, the nutrient-deprived plants wind up shorter than the plants grown in adequate environmental conditions. Thus, finding that the heritability of a characteristic is 100% still does not mean that environmental factors cannot powerfully influence the development of that characteristic.

THE ENVIRONMENTAL FLAW

The math of twin studies is based on the assumption that environments encountered by identical twins are *no more similar* than environments encountered by fraternal twins. However, the formulas are not sound if identical twins, in addition to having more genetic similarity, are also exposed to more environmental similarity; in that case, increased similarities in identical twins' traits can just as easily be accounted for by environmental similarities as by genetic similarities.

In fact, identical twins are exposed to demonstrably more similar environments than are fraternal twins. It starts in the womb: while fraternal twin embryos are always connected to their mother via

two *unique* placentas, identical twins most often (but not always) *share* a single placenta. This means that, beginning soon after conception, identical twins typically have more similar access to nutrients, oxygen, and other factors than do fraternal twins (Figure 3). But does this increased similarity matter? Yes. In fact, identical twins who share a placenta as fetuses have more similar IQs¹⁰ and personalities¹¹ than do *identical* twins who did not share a placenta.

This environmental difference continues after birth. Scarr and McCartney¹² argued that, because similar-looking individuals are more likely than different-looking individuals to evoke similar social responses from people around them, identical twins likely encounter more similar environments than do fraternal twins.

Twin researchers, aware of this essential conjecture underlying their work, have given it a name—the Equal Environments Assumption (EEA)—and have attempted to correct for it in their calculations *and* to demonstrate that it has no real consequences for their findings. Studies have, however, found demonstrable EEA effects^{13–16} that effectively invalidate the assumptions upon which heritability claims rely.

THE BIOLOGICAL FLAW

Most important of all is a deep flaw in an assumption that many people make about biology: That genetic influences on trait development *can be separated* from their environmental context. However, contemporary biology has demonstrated beyond any doubt that traits are produced by *interactions* between genetic and nongenetic factors that occur in each moment of developmental time (see Charney, Genes, behavior, and behavior genetics, *WIREs Cogn Sci*, also in the collection *How We Develop*). That is to say, *there are simply no such things as gene-only influences*. Our DNA, we now know, does not contain specific blueprint-like instructions about traits; rather, DNA segments merely contribute to the production of different kinds of RNA molecules. These RNA molecules can, in turn, regulate other DNA segments, or contribute to the production of proteins that are constituent parts of cells, cells that are assembled into systems that manifest identifiable traits. This entire process takes place in a developmental context; DNA produces its products under the influence of signals from the environment, as well as from other DNA segments (which are in turn signaled by the environment and other DNA segments, and so on). Rather than spitting out pre-determined creations from programmed instructions, 'genes' are more like customized switches that get turned on and

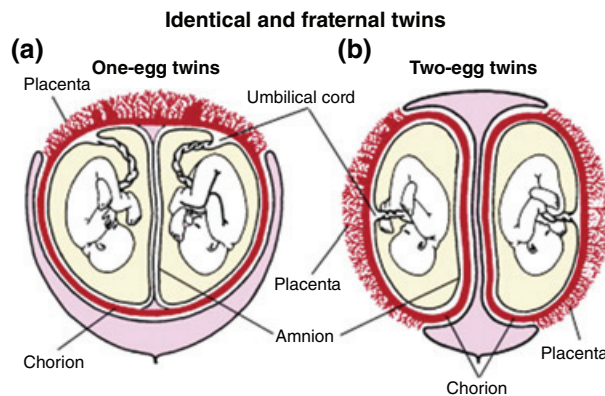


FIGURE 3 | Monozygotic ('identical') twins often share a placenta (a), whereas, dizygotic ('fraternal') twins never share a placenta (b). As a result, most monozygotic twins develop in prenatal environments that are more similar than are the prenatal environments in which dizygotic twins develop. Source: <http://www.britannica.com/EBchecked/topic/217570/dizygotic-twin>.

off by particular developmental circumstances. Traits are *always* a consequence of this interactive dynamic. After determining that intelligence among poor families was nearly zero-percent 'heritable,' Eric Turkheimer and colleagues wrote: 'These findings suggest that a model of [genes plus environment] is too simple for the dynamic interaction of genes and real-world environments during development.'¹⁷

Patrick Bateson has echoed this sentiment, stating that heritability studies make 'the extraordinary assumption that genetic and environmental influences are independent of one another and do not interact. That assumption is clearly wrong.'¹⁸

The fact that DNA segments are influenced by their contexts is one reason that very highly heritable traits can nonetheless be influenced by nongenetic factors. Consider height, which has a heritability that has oftentimes been measured at close to 90% in each of numerous different populations; despite this finding, height can be drastically affected by nutrition, an environmental factor. For example, because food is not equally plentiful in North and South Korea, South Koreans are, on average, nearly five inches taller than North Koreans,¹⁹ even though their gene pools do not substantially differ.²⁰ One likely reason for this is that no gene, or set of genes, transmits isolated growth instructions that directly result in an individual's height. Rather, DNA operates *only* in the context of its particular environment. Like a musician in a band, each DNA segment *only* and *always* plays its part in concert with the other players—in much the same way as a guitarist changes what she does according to the pace of

the drummer, the emotion of the singer, and the charisma of the keyboardist. Similarly, when the exact same DNA segment is operating in a different environmental context, it can generate distinctly different products. It is now clear that nutritional inputs can significantly influence genetic activity, which almost certainly helps explain the height differences found among North and South Koreans. The takeaway message is clear: the now-understood model is not *genes plus environment*, but rather *genes dynamically interacting with the environment*.^{21,22}

WE INHERIT DEVELOPMENTAL RESOURCES, NOT TRAITS

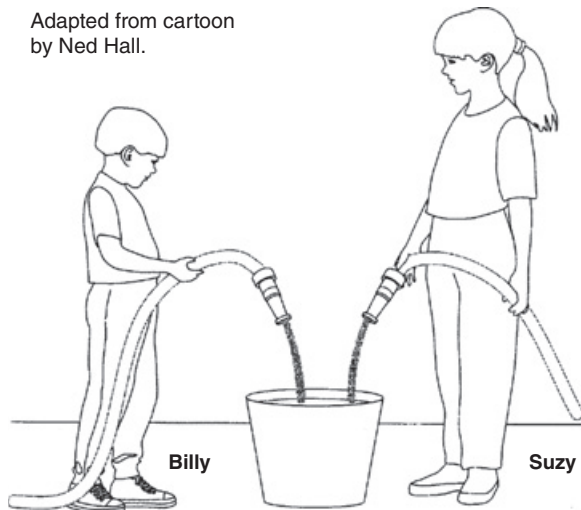
Contrary to popular understanding, people *do not inherit traits* from their parents. Rather, we 'inherit' developmental resources that interact to create the person we each become. These developmental resources include DNA *as well as* nongenetic resources like RNAs, proteins, and the physical, social, and cultural environments in which we develop.^{23–26} These resources range from cytoplasmic factors in the egg to the language spoken in the home.

If DNA does not, in some direct way, determine the development of traits, why do family members often strongly resemble and sound like one another? How do family members 'pass on' diseases, addictions, habits, or personality characteristics? The answer is that inherited genetic variants *do* influence every aspect of our biological and psychological identities, but those influences are mediated and modulated by all of the environmental inheritances that *constantly* interact with those genetic variants. A genetic determinist might look at siblings who share the same parents and say that their physical similarities and differences are due to a mix of shared and non-shared genes. But what we need to understand is that each individual—starting with traits as elemental as eye color—is a developmental creation of genetic and nongenetic factors that are continuously interacting with one another, and that many of the relevant nongenetic factors normally reflect the broader environment outside of our bodies.

One of the ways we know about the profound influences of environments on the formation of traits is through experiments where environmental conditions are altered. For example, when newborn rats developed for 16 days in microgravity aboard the space shuttle Columbia, they matured into adults that exhibited behavioral

1 The bucket model.

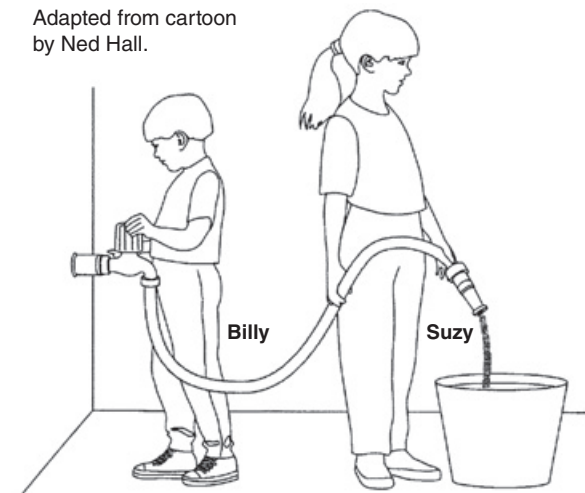
Adapted from cartoon
by Ned Hall.



Here is a bucket: Billy fills it with 40L of water; then Suzy fills it with 60L of water. So, 40% of the water in the bucket is due to Billy, 60% to Suzy.

2 When causes interact.

Adapted from cartoon
by Ned Hall.



But suppose instead that what happened was this: Suzy brought a hose to the bucket, and then Billy turned the tap on. Now how much of the water is due to Billy and how much to Suzy?

Answer: The question no longer makes any sense.

FIGURE 4 | An illustration of why it makes little sense to attempt to quantify the relative importance of two different factors that *interact with one another* to produce an outcome. Because genetic and nongenetic factors interact with one another to produce phenotypes, it is not possible to accurately assess the relative importance of their contributions to the phenotypes they produce. Source: Keller.³⁰

evidence of altered vestibular development.²⁷ Similarly, altering the developmental environment can change what were thought to be the instinctive behaviors of winged-queen fire ants²⁸ (see Blumberg, Development evolving: the origins and meanings of instinct, *WIREs Cogn Sci*, also in the collection *How We Develop*).

CONCLUSION: A TRUE MEASURE OF THE 'INHERITABILITY' OF COMPLEX TRAITS HAS NEVER BEEN DEVELOPED

Although it would, of course, be useful to have a measure of the biological inheritability of complex traits, scientists have never been able to develop such a measure. The process of trait development is so dynamic and multi-causal that in natural contexts where environmental factors are not carefully controlled, accurate predictions about inheritability are effectively impossible. (This stands in contrast to the utility of heritability statistics in experimental and other artificial contexts, in which predictions about a descendant's phenotypes can sometimes be accurate because relevant environmental factors can be precisely controlled; see, for example, Gitonga et al.²⁹)

If a study reveals a trait to be somewhat heritable, we *can* conclude that genetic factors are associated with the trait. But this turns out to be of little value, because we already know that genetic factors influence *all* of our characteristics! At the same time, all of our characteristics are *also* influenced by nongenetic factors that *interact* with the relevant genetic factors, rendering heritability statistics nonsensical in most circumstances (Figure 4).

Heritability statistics do remain useful in some limited circumstances, including selective breeding programs in which developmental environments can be strictly controlled. But in environments that are not controlled, these statistics do not tell us much. In light of this, numerous theorists have concluded that 'the term "heritability," which carries a strong conviction or connotation of something "[in]heritable" in the everyday sense, is no longer suitable for use in *human genetics*, and its use should be discontinued.³¹ Reviewing the evidence, we come to the same conclusion. Continued use of the term with respect to human traits spreads the demonstrably false notion that genes have some direct and isolated influence on traits. Instead, scientists need to help the public understand that all complex traits are a consequence of developmental processes. Without such an understanding, we are at risk of underestimating the

extent to which environmental manipulations can have profoundly positive effects on development.^{32,33} Thus, the way ‘heritability’ is used in most

discussions of human phenotypes not only perpetuates false ideas; it also blinds us to steps we might otherwise take to improve the human condition.

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